1030.01

Industrial Exposure and Control Technologies for OSHA Regulated Hazardous Substances

U.S. Department of Labor
Elizabeth Dole, Secretary
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Volume I of II
Substances A-I

Occupational Safety and Health Administration
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Arsenic and compounds, as As
(CAS NUMBER: 7440-38-2)

SYNONYMS

Arsenic black/Arsenic element/Arsenicals/Arsen (German, Polish) /Colloidal arsenic/Grey arsenic/Metallic arsenic

TRADE NAMES

NONE

DESCRIPTION OF SUBSTANCE

Arsenic is an element with atomic weight 74.92. The most common form of arsenic is a gray brittle crystalline solid with a specific gravity of 5.72. It also exists in amorphous forms: black, specific gravity of 4.7 and yellow, specific gravity of 2.0, which is relatively volatile. Yellow arsenic is soluble in carbon disulfide; the other forms are insoluble in water or solvents, but dissolved by oxidizing acids.

HEALTH EFFECTS

OSHA regulates inorganic arsenic as an occupational carcinogen at 29 CFR 1910.1018. Arsenic compounds are irritants of the skin, mucous membranes, and eyes; arsenical dermatosis and epidermal carcinoma are reported risks of exposure to arsenic compounds, as are other forms of cancer. [PROCTOR AND HUGHES P. 109, 1978]
Cancers resulting from exposure to arsenic usually involve bone structure, and lungs. Despite predominance in the lungs and the face, tumors are also likely to occur in the scrotum, buttocks, abdomen, clavicle, and lower chest. [CASARETT. TOXICOLOGY 1975]
Conjunctivitis produced by inorganic arsenical dusts is characterized by itching, burning, and watering of eyes, with photophobia and sometimes hyperemia and chemosis. [GRANT. TOX OF THE EYE 1974]
Arsenical neuropathy in 57 patients is discussed. Some 37 patients suffered from peripheral neuropathy and 5 from encephalopathy. [JENKINS, RB, INORGANIC ARSENIC AND THE NERVOUS SYSTEM, BRAIN 89: 479 (1966)]
Other chronic effects include generalized itching, sore throat, coryza, lacrimation, numbness, burning or tingling of extremities, dermatitis, vitiligo, alopecia. [GOODMAN. PHARM BASIS THERAP 6TH ED 1980]
Chronic: Bone marrow is seriously injured; all elements of myeloid tissue depressed; aplastic anemia is the hematopoietic disorder most often encountered. [GOODMAN. PHARM BASIS THERAP 6TH ED 1980]
Reported 45 cases of lung cancer and 2 of skin cancer among an unstated number of workers in a refinery. Materials handled were nickel and cobalt ores with substantial arsenic content (range

15-50%). Among personnel not engaged in production, 1 case of cancer was reported. Environment included benzopyrene and sulful dioxide. [IARC MONOGRAPHS. 1972-PRESENT]
Excess incidence of lung cancer in non-ferrous smelter workers; exposure to arsenic was considered to be a contributing factor. [LEE AM, FRAUMENI JF; J. NATL CANCER INST 42 (6): 1045, (1969) AS CITED IN USEPA; AMBIENT WATER QUALITY CRITERIA DOC: ARSENIC P.C-104 (1980) EPA 440/5-80-021]
Survey of 214 babies drinking arsenic-contaminated milk in 1955 in Japan compared to siblings born after. Exposed group had lower IQ, higher incidence of growth retardation, myopia, abnormal EEG, poor dental history, and abnormal facial shape. [USEPA; AMBIENT WATER QUALITY CRITERIA DOC: ARSENIC P.C-52 (1980) EPA 440/5-80-021]
In six cases of chronic arsenic intoxication leukopenia was present with thrombocytopenia in three of them. All had relative eosinophilia. Hematologic abnormalities disappeared within 2-3 weeks after treatment. [KYLE RA; NEW ENGL. J. MED 273: 18 (1965) AS CITED IN NIOSH; CRITERIA DOCUMENT: INORGANIC ARSENIC P.23 (1975) DHEW PUB. NIOSH 75-149]
In Perham, Minnesota, a newly-bored well was associated with illness in 13 people whose hair samples contained arsenic at 37-1,680 ug/g. The well water serving these patients contained arsenic from 11,800 to 21,000 ug/l; this was later determined to come from groundwater contamination by residual arsenical grasshopper bait. [FEINGLASS EJ; NEW ENG J MED 188: 828 (1973) AS CITED IN USEPA; AMBIENT WATER QUALITY CRITERIA DOC: ARSENIC P.C-1 (1985) EPA 440/5-80-021]
Peripheral nervous system effects have been observed in numerous cases of acute, subacute and chronic arsenic exposures. Symptoms include peripheral sensory effects characterized by the appearance of peripheral neuritis symptoms, which originate distally and, over the course of a few weeks, often progressively become more widespread in both lower and upper extremities, usually appearing first in the feet and later in the hands. Signs and symptoms of peripheral motor nerve effects include: Symmetrical muscular weakness of the extremities, predominantly distal but at times extending to proximal muscle groups and, rarely, the shoulder or pelvic girdle; evidence of foot and/or wrist drop; and, in some cases, rapidly developing paralysis and atrophy of lower leg muscles and small muscles of the hand. [USEPA; AMBIENT WATER QUALITY CRITERIA DOC: ARSENIC P.C-63 (1980) EPA 440/5-80-021]
Although arsenic may not act as a direct agent of visceral cancer, there have been many cases of secondary carcinoma of internal organs, e.g., colon, bladder, gallbladder, pancreas, liver, ureter, prostate, lymph nodes, and bronchia as a consequence of metastases of primary skin cancer induced by arsenic exposure (including ingestion, injection, or inhalation). [NAT'L RESEARCH COUNCIL CANADA; EFFECTS OF ARSENIC IN THE CANADIAN ENVIRONMENT P.273 (1978) NRCC NO.15391]
Genitourinary effects: Hematuria, albuminuria, anuria. [ITI. TOX & HAZARD INDUS CHEM SAFETY MANUAL 1982]
Urinary arsenic levels of residents on a downwind transect from a smelter were measured. It was found that arsenic levels

decreased with distance from the smelter. Levels were 0.3 ppm at a distance of 0-0.4 miles and 0.02 ppm at a distance of 2.0-2.4 miles. Samples of vacuum cleaner dust were also collected, and arsenic was reported to decline from 1,300 ppm at a distance of 0-0.4 miles to 70 ppm at a distance of 2.0-2.4 miles. This suggests that arsenic exposure was not confined to one section of the smelter, but extended also to the surrounding community. Thus, the "non-exposed" smelter workers might also have had a degree of arsenic exposure. [MILHAM S JR, STRONG T; ENVIRON RES 7: 176-182 (1974) AS CITED IN NIOSH; CRITERIA DOCUMENT: INORGANIC ARSENIC P.37 (1975) DHEW PUB. NIOSH 79-149]

An outbreak of subacute arsenic poisoning occurred among 9 members of a family; 8 were ill with gastrointestinal symptoms, 4 developed encephalopathy and 2 died. Abnormal liver function tests and leukopenia were common laboratory findings. Epidemiologic and environmental investigations traced the source of arsenic exposure to a farm well with water containing 108 ppm arsenic. The soil adjacent to the well was also contaminated with arsenic, 1 possibly from waste pesticide. Presumably, arsenic gained access to the well through obvious leaks in the well's casing. This was only the second reported outbreak of fatal arsenic poisoning from contaminated drinking water and 1 of few instances where illness followed exposure to a toxic substance which was disposed of, or possibly disposed of, in an indiscriminate manner. [ARMSTRONG CW ET AL; ARCH ENVIRON HEALTH 39 (4): 276-279 (1984)]

Arsenic-associated lung cancers are usually the poorly differentiated type of epidermoid bronchogenic carcinoma. Worker groups with diagnosed lung cancer were studied in copper smelting communities in Montana. Of 25 smelter workers, 4 had well-differentiated epidermoid carcinoma, 10 had poorly differentiated epidermoid carcinoma, 7 had small cell undifferentiated epidermoid carcinoma, and 3 had acinar-type adenocarcinoma. [USEPA; HEALTH ASSESSMENT DOCUMENT: INORGANIC ARSENIC P.7-71 (1984) EPA-600/8-83-021]

Non-fatal skin cancer was found to be related as a function of dose to ingestion of Fowler's solution. Perhaps persons who demonstrate signs of arsenicism following arsenic exposure retain arsenic longer than those who do not show signs, which may suggest that persons who demonstrate signs of arsenicism are at a greater risk of internal malignancies. This was suggested by the fact that all of the deaths from internal malignancies in the subset of patients given Fowler's solution who were examined for signs of arsenicism occurred in individuals who demonstrated such signs. [USEPA; HEALTH ASSESSMENT DOCUMENT: INORGANIC ARSENIC P.7-73 (1984) EPA-600/8-83-021]

Airborne metallic particulates are associated with fossil-fueled power plants, automobile exhausts, metal mining and metallurgical smelters. The possible toxic effects of metals on the lung are of environmental and occupational concern. In this investigation the effects of in vitro exposure to metallic ions on the following parameters were determined: O2 consumption and membrane integrity of alveolar macrophages and Type 2 cells, and chemiluminescence of zymosan-stimulated alveolar macrophages. Short-term in vitro exposure to pentavalent asenic had little

effect on alveolar macrophages and Type 2 cells. Although the data suggested that exposure to certain metals may have been harmful to the lung, the pulmonary parameters tested in this investigation displayed differing susceptibility to metal exposure. Metals were less toxic to alveolar Type 2 cells than to alveolar macrophages. Data also indicated that chemiluminescence was the most sensitive assay for monitoring the viability of alveolar macrophages, while O2 consumption was a sensitive assay for Type 2 cells. [CASTRANOVA V ET AL; J TOXICOL ENVIRON HEALTH 13 (4-6): 845-856 (1984)]
The effects of trivalent and pentavalent arsenic on the synthesis of DNA, RNA, and protein in cultured human cells were compared. The clastogenicity of these compounds were also compared. The chromosome-breaking activity in cultured (human) leukocytes was significantly higher for the compound with trivalent than with pentavalent arsenic. The activity in cultured human skin fibroblasts was similar to that in leukocyte cultures. The colony-forming capacity after exposure to arsenic compounds indicated that trivalent compounds were more toxic than pentavalent compounds. Both trivalent and pentavalent arsenic inhibited DNA and protein synthesis in leukocytes. [NAKAMURO K, SAYATO Y; MUTAT RES 88 (1): 73-80 (1981)]
Excess skin cancer and a gangrenous condition of the hands and feet, called Blackfoot's disease, was found in people in Taiwan due to arsenic in drinking water. [TSENG W; ENVIRON HEALTH PERSP. 19: 109, (1977) AS CITED IN USEPA; AMBIENT WATER QUALITY CRITERIA DOC: ARSENIC P.C-57 (1980) EPA 440/5-80-021]

TOXICITY/EXPOSURE LIMITS

NFPA RATING - NONE

TOXICITY HAZARD RATING - Acute and chronic local: skin 3; mucous membranes 3; eyes 3. Acute and chronic systemic: ingestion 3; inhalation 3; skin 3. 3= High: may cause death or permanent injury after exposure to small quantities. [SAX. DANGER PROPS INDUS MATER 6TH ED, P. 316, 1984] 5.5= Extremely toxic: probable oral lethal dose (human) 5-50 mg/kg, between 7 drops and 1 teaspoonful for a 70 kg person (150 lbs.). [GOSSELIN. CTCP 5TH ED, P. II-129, 1984]

IMMEDIATELY DANGEROUS TO LIFE OR HEALTH - NONE - Carcinogen

OSHA PEL -	******* ppm,	0.500	mg/m3;TWA -	as Arsenic (organic)
	******* ppm,	0.010	mg/m3;TWA -	as Arsenic (inorganic)

See 1910.1018(a) for applications excluded.

ADOPTED ACGIH/TLV - ****** ppm, 0.200 mg/m3;TWA - as Arsenic (soluble compounds)

NIOSH/REL - ****** ppm, 0.002 mg/m3;STEL - 15 minutes Human carcinogen (inorganic)

INDUSTRY USE DATA

Arsenic and its compounds are used in the manufacture of certain types of glass; in metallurgy for hardening copper, lead alloys. [MERCK INDEX 9TH ED 1976]
Used in herbicides, insecticides, rodenticides and paints. [GOSSELIN. CTCP 4TH ED 1976]
To control sludge formation in lubricating oils; [PATTY. INDUS HYG & TOX 2ND ED VOL2 1963]
To make gallium arsenide for dipoles and other electronic devices; special solders of high-purity. [HAWLEY. CONDENSED CHEM DICTNRY 9TH ED 1977]
Doping agent in germanium and silicon solid state products of high-purity. [HAWLEY. CONDENSED CHEM DICTNRY 9TH ED 1977]
Component of alloys; component of electrical devices. [SRI]
Taxidermy and tanning. [NIOSH; CRITERIA DOCUMENT: INORGANIC ARSENIC P.14 (1975) DHEW PUB. NIOSH 75-149]
Used as a growth stimulant for plants and animals. [USEPA; AMBIENT WATER QUALITY CRITERIA DOC: ARSENIC P.A-1 (1980) EPA 440/5-80-021]
Constituent of antifungal and weather-resisting wood treatments. [KRZYZEWSKI J; CAN PATENT NO. 1174004 (9/11/84)]
To manufacture arsenical organic compounds for therapeutic use. [BROWNING. TOX INDUS METALS 2ND ED 1969]

NIOSH 1982 NATIONAL OCCUPATIONAL EXPOSURE SURVEY

SIC CODE	INDUSTRY NAME	TOTAL ON PAYROLL	TOTAL EXPOSED	PERCENT EXPOSED
3332	PRIMARY LEAD	300	191	63.67

NIOSH 1972 NATIONAL OCCUPATIONAL HAZARD SURVEY

SIC CODE	INDUSTRY NAME	TOTAL ON PAYROLL	TOTAL EXPOSED	PERCENT EXPOSED
3211	FLAT GLASS	925	48	5.19

OSHA/EXPOSURE DATA

NONE

ENGINEERING CONTROLS

General ventilation; local exhaust ventilation; hood; enclosure of process or worker, as needed; air cleaning equipment.

PERSONAL PROTECTIVE EQUIPMENT

Protective clothing, gloves, goggles, and a hood for head and neck should be provided. [SITTIG M; HANDBOOK OF TOXIC AND HAZARDOUS CHEMICALS P.60 (1981)]
Wear boots and long-sleeved coveralls. [ITI. TOX & HAZARD INDUS CHEM SAFETY MANUAL 1982]
Respiratory protection should be as follows: At any detectable concentration: any self-contained breathing apparatus with full facepiece and operated in a pressure-demand or other positive pressure mode or any supplied-air respirator with a full facepiece and operated in pressure-demand or other positive pressure mode in combination with an auxiliary self-contained breathing apparatus and operated in pressure-demand or other positive pressure mode. Escape: any air purifying full facepiece respirator (gas mask) with a chin-style or front- or back-mounted acid gas canister having a high-efficiency particulate filter or any appropriate escape-typeself-contained breathing apparatus. [NIOSH: POCKET GUIDE TO CHEMICAL HAZARDS P. 55 (1987) DHEW (NIOSH) PUB NO. 85-114]

STORAGE

Protect against physical damage. Store in a cool, dry location. Separate from food, oxidizing agents, and acids. [NFPA, P. 49-19, 1986]